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DEXMEDETOMIDINE FOR ATTENUATION OF PRESSOR RESPONSE OF LARYNGOSCOPY AND INTUBATION

Anaesthesiology

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ABSTRACT

The attendant danger of hypertension and tachycardia observed during laryngoscopy and intubation, needs considerable attention to prevent their consequences such as arrhythmia and myocardial ischemia. In patients with cardiovascular disease these hemodynamic changes may lead to life threatening complications like acute heart failure, cerebrovascular accidents. The aim of our study is to evaluate the efficacy of IV dexmedetomidine premedication on attenuation of haemodynamic response (HR and MAP) to laryngoscopy and intubation.

KEYWORDS

α2 adrenoreceptor, sympathoadrenal response, tracheal intubation.

Introduction:

Laryngoscopy and tracheal intubation causes intense autonomic reflex responses consisting of increased circulating catacholamines, tachycardia, hypertension, myocardial oxygen demand, and dysarrythmias. To obtund haemodynamic response lignocaine, opiods, nitroprusside, nitroglycerine, vearpamil, nifedipine, esmolol, clonidine and recently, dexmedetomidine have been studied.

The attendant danger of hypertension and tachycardia observed during laryngoscopy and intubation, needs considerable attention to prevent their consequences such as arrhythmia and myocardial ischemia. In patients with cardiovascular disease these hemodynamic changes may lead to life threatening complications like acute heart failure, cerebrovascular accidents.[1] To obtund the cardiovascular responses various drugs used are lignocaine in various forms, high doses of opioids like fentanyl, alfentanyl, sufentanyl, buprenorphine etc, vasodilators like sodium nitroprusside, nitroglycerine, hydralazine, calcium channel blockers like verapamil, nifedipine,[2] nitradepine, manidepine etc, alpha adrenergic blocker droperidol, beta adrenergic blockers metoprolol, esmolol,[3] magnesium sulphate,[4] alpha adrenergic agonist like clonidine[5] and recently dexmedetomidine. [6] However, no modality is devoid of drawbacks and limitations. Dexmedetomidine, a highly selective α-2 adrenergic agonist has sedative and analgesic effects[7,8] and produces hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus cerelous leading to decreased systemic noradrenalin release that results in attenuation of sympathoadrenal responses and hemodynamic stability during laryngoscopy and tracheal intubation. [9] The aim of our study is to evaluate the efficacy of IV dexmedetomidine premedication on attenuation of haemodynamic response (HR and MAP) to laryngoscopy and intubation.

Materials and Methods:

This study was done in the Department of Anesthesia, Azeezia Institute of Medical Sciences, Kerala.

This study was done from June 2017 to May 2018.

In this double-blind, randomized, controlled clinical trial, after approval from institutional ethical committee, and with written informed consent, eighty normotensive patients of either sex, aged 25-65 yrs, ASA class I or II scheduled for elective surgeries to be performed under general anaesthesia (GA) requiring endotracheal intubation, were recruited. Patients were excluded if had body weight more than 150% of their ideal body weight using Broca"s index, had Contraindication to study drug, with obvious intubation difficulty, Pregnant or lactating patient, Hypertensive patients receiving any antihypertensive, had Psychiatric disorders. Randomization was done by computer generated randomization table using Software "Minitab", random sequence was generated by random allocation software. Patients were allocated in 2 groups; Group D (n=40)to receive single bolus IV dose of dexmedetomidine (0.6 µgkg-1) diluted upto 20 ml as pre-medication or Group C (n=40) to receive IV saline (20 ml) as pre-medication. Syringe containing pre-medication (either dexmedetomidine or normal saline) was prepared by a team member who was not involved in the data recording. Intraoperative monitoring included three lead ECG, plethysmographic pulse oximetre, capnometry, non-invasive arterial pressure was performed. The observations were made by the same observer in all the patients so as to avoid observer bias. Patients were pre-medicated with a single dose of dexmedetomidine 0.6µgkg-1 IV using 20ml syringe pump over 10 min in group D and the same amount of saline was given to the patient in the control group. Both the groups were also premedicated with IV fentanyl 1µgkg-1. After pre-oxygenation for 3 min both groups were induced with injection thiopental 5 mgkg-1 and Succinylcholine 2 mgkg-1 was administered. After ceasation of fasciculations gentle laryngoscopy was done by trained anaesthesiologist and patient was then intubated with proper size cuffed endotracheal tube under direct vision. Correct position of tracheal tube was verified by auscultation of chest and by capnometry. It was decided to exclude those patients from study who require more than one attempt for intubation. However all the patients in our study were intubated in first attempt and there was no exclusion. Intraoperative lactated Ringer solution was administered at 4mlkg-1 h -1 iv. Further management of cases was done according to institutional protocol for general anaesthesia. Hypotension (MAP ≤30% from baseline) was treated with IV ephedrine 6 mg and bradycardia (heart rate ≤30% from baseline) was treated with IV glycopyrolate 0.2 mg. Mean arterial pressure (MAP), heart rate (HR) readings were taken at following time interval: • T1: Baseline on OT table • T2: 10 min after pre-medication. • T3: 30 seconds after thiopental. • T4: 30 second after succinylcholine at laryngoscpy. • T5: immediately after intubation. •T6: at 1 min after intubation. • T7: at 3 min after intubation • T8: at 5 min after intubation.

Results:

- With regard to age, weight, gender, MPC grading, and baseline HR, MAP there were no significant differences between two groups.
- There was statistically significant initial fall in HR in group D after dexmedetomidine (p=0.000).
- There was rise in HR, after laryngoscopy and intubation, remained raised for 3 min postintubation in both the groups (p=0.000 & 0.017).
- But this rise was statistically significantly more in control group from T2-T6 as compared to group D (p=0.000; 0.001; 0.003; 0.000; 0.0140 at various levels from T2-T6).
- Heart rate in both groups was almost near to the baseline values at T7 and T8 (p=1.000).
- The MAP was increased significantly compared with preoperative value after intubation in the group C (p=0.000) and was significantly higher than in group D (p=0.000).
- In the group D, MAP was not significantly higher than the preoperative value at all times.
- No incidence of hypotension or bradycardia requiring intervention was reported in both groups.

Discussion:

We observed highly significant rise in MAP at laryngoscopy and immediately after intubation ($p < 0.05$) in control group. Dexmeditomidine group showed significant fall after giving study drug, it remained status co and there was no rise after laryngoscopy & intubation. As compared to control group. This observation is in agreement with previous studies.[6,10,11]

HEART RATE: There was significant rise in heart rate control group starting from T2 till T5, but it was more pronounced at T4 and T5 i.e., After laryngoscopy and intubation ($p < 0.05$). Dexmeditomidine group showed initial fall and then significantly less rise after laryngoscopy and intubation as compared to control group. This observation is in agreement with previous studies.[6,11.]

Conclusion:

I.V dexmedetomidine 0.6µg premedication is advantageous as it is found to be effective and beneficial in attenuating the haemodynamic response of laryngoscopy and intubation to prevent its consequences.

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